the fairy dart. Do not forget that when you sneeze, the fairies are endeavouring to carry you off, and that if a sudden lameness overtakes you, it indicates that you have been trespassing on fairy ground. The great safeguards against the fairies are fire, iron, and dung, the last-mentioned because the fairies, being pure spirits, cannot bear defilement. Never take fire out of a house with a person sick within its walls, or misfortune will surely attend you.

Do not blame the fairies for all your ailments, for you may have been looked upon by someone possessing the evil eye. If you should meet anyone whom you know of a certainty to possess the evil eye, be sure to double your thumbs under your fingers, and if by any unfortunate chance he should praise anything or anyone belonging to you, never lose sight of him until, by whatever form of strategy you please, you have persuaded him to spit, so that you may annoint the object of his admiration with it, and so avert sickness or other misfortune.

Dublin Journal of Medical Science, 1881.

Proceedings of the American Philosophical Society, 1887.

"Lectures on the MS. Materials of Ancient Irish History." Prof. O'Curry.

Still-born Infants

By RICHARD H. HUNTER, M.D., M.CH., PH.D., M R.I.A.

Queen's University, Belfast

THE problem of the still-born infant is one of primary importance to the practitioner, but as a general rule many possible causes of the condition are overlooked, and the practitioner is left wondering why a still-born infant should have resulted from what had been an apparently normal labour. This is possibly due to the fact that many people do not properly appreciate the vast difference between death in an organism which is *fully* alive as a separate entity, as a child or adult, and death in an organism which has not yet reached the state of a separate existence, as a feetus.

The difference in the two types of death may be illustrated by the two methods of respiration employed before and after birth. After birth, respiration is a gaseous exchange between the atmospheric air in the lung alveoli and the blood as it circulates in the lung tissue. Before birth, respiration is a gaseous exchange between two liquids, the maternal blood and the fœtal blood, as they circulate in the uterine sinusoids and in the placental villi. This remarkable difference in the method of respiration is of great importance in discussing how a fœtus dies of intra-uterine suffocation.

This obvious difference in the physiology of pre-natal and post-natal life must be remembered in examining a still-born infant; and the difference in other physiological processes must also be borne in mind, such as the difference between antenatal and post-natal nutrition, excretion, secretion, etc.

It is obvious that the causes of still-born infants must vary with the stage of delivery the infant has reached at the time death occurs, and there is a wide range of factors responsible. But in this short paper it is proposed to confine our observations to those factors which show no clear external signs of the lesion responsible. These may be divided conveniently into circulatory disturbances, injuries, and congenital deformities.

CIRCULATORY DISTURBANCES.

Fœtal respiration is dependent on the existence of an efficient placental circulation, and any interference with the placenta or umbilical cord will result in a greater or lesser disturbance of the fœtal metabolism. Such a disturbance may manifest itself in one of two ways: (a) An undue rise in the hydrogen-ion concentration of the blood, or (b) in a lack of oxygen in the blood.

- (a) If an undue rise in the hydrogen-ion concentration occurs, this will cause a stimulation of the vagal centres in the medulla, with a consequent slowing in the heart-beat and a stimulation of respiration. Such premature efforts at respiration, while the fœtus is floating in amniotic fluid, will cause an intake of the fluid into the respiratory passages, and when the infant is born it is unable to draw air into its lungs, and it is to all intents and purposes drowned in its own amniotic fluid.
- (b) If a lack of oxygen results, this will express itself on the medulla, where the centre of respiration may become damaged to such an extent that its power of response to the ordinary post-natal stimuli becomes lost, and the infant fails to breathe at birth.

The normal change from intra-uterine to pulmonary respiration is dependent upon the reflex response of a healthy medullary centre. If the infant is born with its heart beating, it is alive, and a failure to breathe means either failure in medullary response, or some abnormality of the respiratory apparatus, making respiration impossible.

The respiratory centre fails to respond when it is severely damaged by prolonged oxygen-lack. In some cases artificial respiration may oxygenate the blood sufficiently to lead to recovery of the centre, but in other cases the damage may be so severe that all efforts are unavailing, and there are no naked-eye appearances found at the post-mortem examination to account for the infant's death.

It is commonly stated that placental infarction and hæmorrhage (two common causes of circulatory disturbances in the fœtus) are due to syphilis, but this view is no longer held. The syphilitic placenta may appear normal to the naked eye, but in cases where the pathological changes are well marked, the placenta is pale and somewhat bulky looking, and the cut surface is strikingly bloodless. Microscopically the main changes are in the villi. Normally these consist of thin-walled capillaries, loose stroma, and a covering of epithelium one or two layers thick, depending on the period of gestation. In the syphilitic placenta the fine capillaries fail to grow, and are either absent or are but poorly developed in many villi. The villi are much increased in bulk, as the stroma has grown enormously, and there is usually a well-marked round-cell infiltration. The effect of this increase in size of the villi

is to decrease the intra-villous spaces. The blood circulation in the villi (fœtal) and around the stroma (maternal) is thus enormously decreased, with a resultant deficiency in the nourishment and æration of the fœtal tissues. These changes in the placenta may cause the changes already described in the medulla, and which result in a still-born infant.

Another cause of circulatory disturbance is infarction of the placenta. These are said to be due, primarily, to a clotting of the blood in the intervillous spaces of the placenta. This is possibly due to a toxin circulating in the maternal blood-stream, producing a stagnation of the blood, with coagulation around the villi. Fibrin is deposited on the villi, which, being deprived of their nourishment, undergo necrosis. The clot organises around the damaged villi, and finally the villi appear as mere strands in a mass of organised clot, the whole being an infarct.

The external signs of suffocation in a still-born infant are lividity of the general body-surface, congestion of the conjunctiva, and blackness of the tips of the fingers and the nail-beds. Post-mortem rigidity is usually absent, owing to delay in the clotting-time of the blood, caused by its high CO₂ content.

The most importance evidence of suffocation is found in the thorax, as well-marked sub-pleural and superficial hæmorrhages. Hæmorrhages may also be found under the capsule of the thymus, on the surface of the aorta and on the pulmonary artery, and under the parietal pleura. The auricles of the heart are usually engorged. The vessels of the pia mater and those of the brain substance are intensely engorged. The abdominal organs may show no abnormal signs, but they are usually congested. The trachea in some cases contains a thin frothy fluid, which is taken as evidence of premature efforts at breathing. The blood is in almost all cases very dark and very fluid, owing to the high CO₂ content.

CRANIAL INIURIES.

One of the commonest causes of still-born infants, and one which presents no external signs of the causation, is hæmorrhage within the cranium, due to a tear in the neighbourhood of the falx cerebi and the tentorium cerebelli. When a tear occurs in this region, the hæmorrhage spreads evenly over the surface of the occipital lobe of the cerebrum, over the temporal lobe, and may even reach, in an upward direction, the median fissure, and may even spread over the upper surface of the cerebellum.

Eardly Holland, in his Report to the Ministry of Health some few years ago, gave the following list of what he calls "primary factors" responsible for tentorial tears:—

- (a) Breech deliveries.
- (b) Forceps application.
- (c) Rapidity of the second stage of labour.
- (d) Prolongation of the second stage of labour.
- (e) Contracted pelvis.

Tentorial tears, according to this authority, are due to an antero-posterior stress on the head, which stretches the falx cerebi upwards and tears the vertical fibres. "Decrease in antero-posterior measurements is brought about by pressure applied at opposite ends of the diameters lying between the occiput and forehead, i.e., sub-occipit-frontal, and occipito-frontal; such pressures occur during labour in a vertex presentation, and are correspondingly increased when the pelvis is contracted."

Holland assumes that the production of the tears occurs either when the delivery of the head is so sudden that no time for moulding is allowed; in rapid extraction of the after-coming head in breech presentations; and in precipitate labours, or when the external force used in forceps deliveries is wrongly applied.

CONGENITAL DEFORMITIES.

Certain internal congenital deformities result in still-born infants, e.g., extreme cases of diaphragmatic hernia, achondroplasia, polycystic disease of the kidneys, or absence of kidneys.

The condition of congenital diaphragmatic hernia is certainly much more common than generally supposed, for most cases pass unrecognised, as post-mortem examinations are not made. There are in the literature about 168 cases described, and of these 91 were still-born infants, 55 lived for a few minutes to a few hours, and 20 lived for periods of a few days to a few weeks.

Death is due to large portions of the intestine, together with the stomach and a greater or lesser portion of the liver, passing through an abnormal congenital opening in the diaphragm, filling the thoracic cavity and preventing the expansion of the lungs after birth.

Achondroplasia may, and often does, cause death in the new-born by premature ossification and fusion of the occipital elements around the foramen magnum. The spinal cord as it develops is pressed upon by the hard bony margins of the foramen, and eventually "nips" and may even cut right through the cord, cutting off the transmission of respiratory impulses along the nerve-tracts. As a result the infant fails to respond to the time-honoured slap, and is still-born.

It is a little more difficult to express in a few words the mode of death in cases of complete polycystic disease of the kidneys, or in absence of the kidneys, without reference to the first portion of these notes. When the fœtus is still alive within the uterus, the waste products of metabolism are excreted through the placenta into the maternal blood, but when the infant is born, all waste products must be excreted through the kidneys. If these are not present, or not properly developed, the waste products accumulate within the blood-stream of the infant and produce toxemia and death. This sequence of events can be readily understood when the infant lives for a few days after birth. But the writer of these notes has seen no less than five cases of still-born infants in which the most careful post-mortem examination revealed nothing but an absence of both kidneys in three cases, and complete polycystic disease in the other two.